Clean Version of All Claims under Examination
(claims unamended herewith appear in small font)

1. (Amended) A method for diagnosing, or determining a predisposition to developing, an arterial wall disruptive disorder in a subject, comprising:

perform a radiological or anatomically targeted procedure on the subject to detect a symptom indicative of arterial wall disruptive disorder;

perform an ophthalmological procedure on the subject to detect presence of drusen; and

detecting one or more genotypic or phenotypic markers for macular degeneration;

whereby the subject is diagnosed to have an arterial wall disruptive disorder or a predisposition to developing an arterial wall disruptive disorder.

2. The method of claim 1, wherein said arterial wall disruptive disorder is selected from the group consisting of: an aortic aneurysm, a peripheral aneurysm, a visceral aneurysm, and an intracranial aneurysm.

3. The method of claim 1, wherein said arterial wall disruptive disorder is a dissecting aneurysm.

4. The method of claim 2, wherein said aortic aneurysm is an abdominal aortic aneurysm (AAA).

5. The method of claim 2, wherein said aortic aneurysm is a thoracic aortic aneurysm (TAA).

6. The method of claim 1, wherein said macular degeneration is age-related macular degeneration (AMD).

7. The method of claim 1, wherein said macular degeneration is the exudative or neovascular (wet) form, which is characterized by disciform scars and/or choroidal neovascularization (DS/CNV) or an exudative precursor phenotype.

9. The method of claim 1, wherein said marker is one or more drusen-associated markers.

10. The method of claim 9, wherein said drusen-associated marker is selected from the group consisting of immunoglobulins, amyloid A (α1 amyloid A), amyloid P component, C5b-9 terminal complexes, HLA-DR, complements 3, 5 and 9, complement reactive protein (CRP), immunoglobulin lambda and kappa light chains, Factor X, HLA-DR, apolipoprotein A, apolipoprotein E, antichymotrypsin, β2 microglobulin, fibrinogen, prothrombin, thrombospondin, elastin, collagen, vitronectin, ICAM-1, LFA1, LFA3, B7, IL-1, IL-6, IL-12, TNF-alpha, GM-CSF, heat shock proteins, colony stimulating factors (GM-CSF, M-CSFs), TNFα, and IL-10.

12. The method of claim 9, wherein said drusen-associated marker is a phenotypic marker selected from the group consisting of RPE cell death or dysfunction, immune mediated events, dendritic cell proliferation, dendritic cell migration, dendritic cell differentiation, dendritic cell maturation and activation in the sub RPE space, the presence of disciform scars, the presence of choroidal neovascularization, and the presence of choroidal fibrosis.

19. The method of claim 12, wherein said fibrosis is detected by determining the presence or level of elastin, fragments of elastin, collagen, or fragments of collagen.

---

36. (Twice Amended) A method for diagnosing, or detecting a predisposition to developing, an arterial wall disruptive disorder in a subject, comprising:

perform a radiological or anatomically targeted procedure on the subject to detect a symptom indicative of arterial wall disruptive disorder;

C 2

perform an ophthalmological procedure on the subject to detect presence of drusen; and

performing an immunoassay on a sample obtained from said subject to detect a gene product indicative of macular degeneration;

whereby the subject is diagnosed to have an arterial wall disruptive disorder or a predisposition to developing an arterial wall disruptive disorder.